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Atypical HSV encephalitis infection: A Zebra

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Introduction

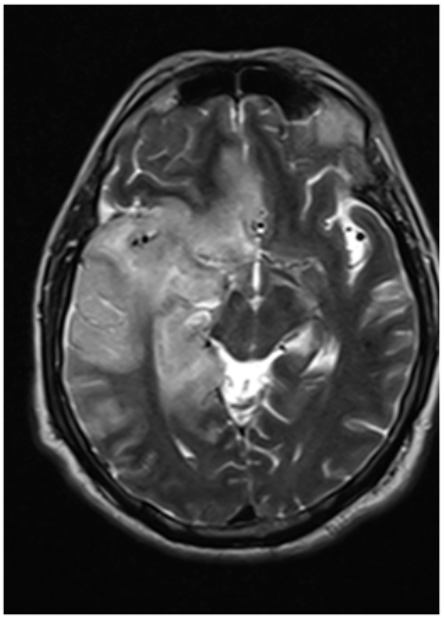
HSV encephalitis is a well known entity in the world of neurology. It is well known that if not treated in time it is a potentially fatal and debilitating disease. Here we discuss an atypical presentation as well as rare imaging features associated with this infection.The aim of this case report is to remind the reader that even infectious process can present like acute vascular syndromes and highlight the imaging findings not typically associated with this processso timely diagnosis can be made and lives be saved.

Keywords

HSV encephalitis; Atypical infection; Rare radiological findings

Case History

We present a case of a 52 year old man in apparently good health presenting with sudden onset of left sided weakness and altered mental status. He had been doing his usual work as a laborer the day before and had no preceding trauma, viral prodrome, fever or rash. In the emergency room an urgent head CT was done which was unremarkable. This was followed by a MRI and MR angiogram scan of the brain which showed right hemispheric diffusion restriction in the territory of the right middle cerebral artery and right anterior cerebral artery. The vascular imaging was unremarkable. The patient was admitted to the neuro ICU where a guarded lumbar puncture was performed and was suggestive of viral meningitis with high protein and lymphocytosis. There were no red blood cells and glucose was normal. He was empirically started on acyclovir and CSF PCR for herpes virus was sent which came back positive at a later date. While in the ICU the patient had an EEG which showed generalized slowing without any evidence of seizures. His mental status and weakness improved over the next two days when he was shifted out of the ICU and then subsequently discharged without any deficits. (Figure1)



T2

Figure 1: **T2** sequence showing right frontal, temporal and deep white matter involvement.

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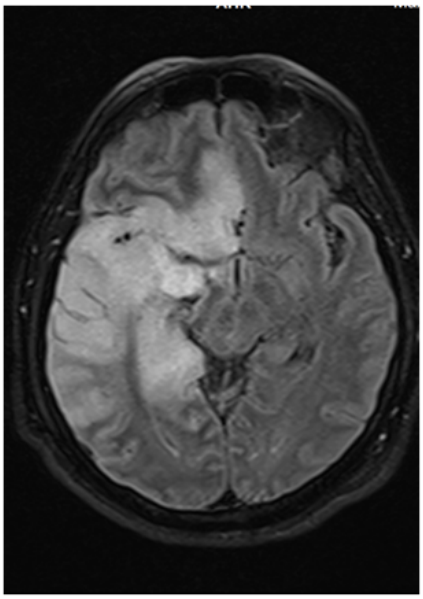
Discussion

Herpes Simples Viral Encephalitis (HSE) if one of the most common and fulminant viral infections. If not treated in time it can cause reversible neurological damage. Most commonly HSV-1 infection causes viral encephalitis. Patients usually present with amnesia, confusion, fever, headache or seizures.The onset of symptoms is rapid over the course of a few days [1]. HSE tends to affect patients younger than 20 and older than 50 years of age and both sexes are at equal risk of contracting the disease [2]. Sometimes focal neurological signs may develop and patients may have hemiparesis and difficulty talking. HSE can leave the patient with permanent neurological deficits hence prompt diagnosis and early empirical treatment is preferred to limit focal neurological damage. Diagnosis of HSE is based on clinical findings, cerebrospinal fluid examination and radiological studies. Acute HSE CSF has a typical profile of viral infection but, sometimes red blood cells (10-500/uL) and xanthochromia may be seen. CSF Protein (60-700 mg/dL) is elevated and CSF glucose may be normal or slightly decreased [3]. CSF PCR for HSV1 and HSV2 remains the main stay for diagnosing HSV. CSF PCR has a sensitivity of 94-98% and specificity of 98-100%. CSF PCR turns positive within 24 hours of the onset of symptoms and can remain positive for at least 5-7 days after the start of antiviral therapy. The test may be falsely negative in the early phase of infection (<72 hours of onset of symptoms) and may need to be repeated if the clinical suspicion is high [4]. MRI is the imaging modality of choice and HSV infection. Typical of HSE is bilateral asymmetrical involvement of the limbic system, medial temporal lobes, insular cortices and inferolateral frontal lobes. The basal ganglia are typically spared [5]. Sometimes hemorrhagic changes may also be seen on the MRI. Unilateral hemispheric involvement in HSE is very rare. As in our case there was unilateral involvement of the right hemisphere and the cingulate cortex with the involvement of basal ganglia. There are very few case reports in the literature with such extensive and unilateral involvement in HSE [5]. As discussed above the patients usually present with a rapid decline over the course of few days however, in our case the presentation was more like an acute vascular event which prompted the workup on the lines of stroke. Once a large vessel occlusion was ruled out a differential diagnosis of viral encephalitis was made based on clinical findings, CSF studies and the imaging. It is both interesting and essential to be aware of such a presentation of treatable viral encephalitis which carries a high mortality and morbidity. Our patient was treated with a 14 days course of acyclovir and discharged without any deficits. (Figure 2-4)

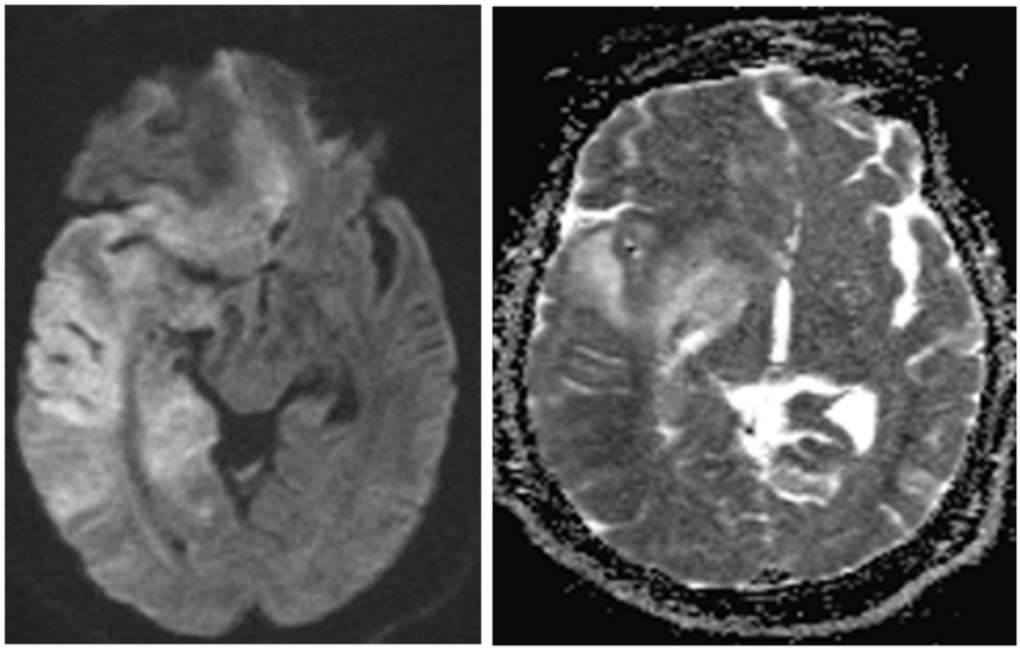


Flair

Figure 2: FLAIR sequence showing hyperintensities in the corresponding area.

Conclusion

Even infectious neurological disorders can present like an acute vascular syndrome. Also, the atypical radiological features of the HSV infection may simulate a vascular syndrome and mislead a clinician. It is important to be aware of such a presentation while treating patients with acute neurological deficits.



DWI **ADC**

Figure 3 & 4: DWI and ADC shows diffusion restriction without ADC correlate.

References

1. Whitley RJ, Cobbs CG, Alford CA Jr, Soong SJ, Hirsch MS, et al. Diseases that mimic herpes simplex encephalitis. Diagnosis, presentation, and outcome. NIAD Collaborative Antiviral Study Group. JAMA. 1989 Jul. 262:234-239.
2. Whitley RJ, Soong SJ, Linneman C Jr, Liu C, Pazin G, et al. Herpes simplex encephalitis. Clinical Assessment. JAMA. 1982 Jan. 247:317-320.
3. Mook-Kanamori B, van de Beek D, Wijdicks EF. Herpes simplex encephalitis with normal initial cerebrospinal fluid examination. J Am Geriatr Soc. 2009 Aug. 57:1514-1515.
4. Domingues RB, Lakeman FD, Mayo MS, Whitley RJ. Application of competitive PCR to cerebrospinal fluid samples from patients with herpes simplex encephalitis. J ClinMicrobiol. 1998 Aug. 36:2229-2234.
5. Bulakbasi N, Kocaoglu M. Central nervous system infections of herpesvirus family. Neuroimaging Clin. N. Am. 2008;18: 53-84.